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Inflammation following acute myocardial infarction: Multiple players, dynamic roles, and novel therapeutic opportunities

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ABSTRACT

Acute myocardial infarction (AMI) and the heart failure that often follows, are major causes of death and disability worldwide. As such, new therapies are required to limit myocardial infarct (MI) size, prevent adverse left ventricular (LV) remodeling, and reduce the onset of heart failure following AMI. The inflammatory response to AMI, plays a critical role in determining MI size, and a persistent pro-inflammatory reaction can contribute to adverse post-MI LV remodeling, making inflammation an important therapeutic target for improving outcomes following AMI. In this article, we provide an overview of the multiple players (and their dynamic roles) involved in the complex inflammatory response to AMI and subsequent LV remodeling, and highlight future opportunities for targeting inflammation as a therapeutic strategy for limiting MI size, preventing adverse LV remodeling, and reducing heart failure in AMI patients.

Contents

Abbreviations: ACS, Acute coronary syndrome; AMI, Acute myocardial infarction; AGEs, Advanced glycation end-products; BAFF, B-cell activating factor; C1-INH, C1-inhibitor; CCL2, Chemokine ligand 2; CCL5, Chemokine ligand 5; CCR2, Chemokine receptor 2; CCR5, Chemokine receptor 5; CCR9, Chemokine receptor 5; CR1, Complement receptor 1; CINC-1, CXCL1, GRO α, KC, Cytokine-induced neutrophil chemoattractant 1; DAMPs, Damage-associated molecular patterns; ECM, Extracellular matrix; EDA, Extra domain A; eRNA, Extracellular ribonucleic acids; FN-EDA, Fibronectin-end domain A; HSPs, Heat shock proteins; hs-CRP, High-sensitivity C-reactive protein; HMGB1, High mobility group box 1; ICAM-1/ICAM-2, Intercellular adhesion molecule; IFN-γ, Interferon-γ; IRF5, Interferon regulatory factor 5; IHD, Ischemic heart disease; IL-1, Interleukin-1; IL-8, CXCL8, Interleukin-8; LV, Left ventricular; LTB4, Leukotriene B4; MIP-2α, CXCL2, GRO β, Macrophage inflammatory protein-2α; MMPs, Matrix metalloproteinases; MCP-1, Monocyte chemoattractant protein-1; MyD, Myeloid differentiation primary response gene; MI, Myocardial infarction; NO, Nitric oxide; NLRs, NOD-like receptors; NF-κB, Nuclear factor kappa-light-chain-enhancer of activated B cells; NLRP3, Nucleotide-binding oligomerization domain-like receptor family of cytosolic proteins; PRRs, Pattern recognition receptors; PS, Phosphatidylserine; PMN, Polymorphonuclear leukocytes; PPCI, Primary percutaneous coronary intervention; ROS, Reactive oxygen species; RAGE, Receptor for advanced glycation end-products; Tregs, Regulatory T cells; STEMI, ST segment elevation myocardial infarction; TLRs, Toll-like receptors; TGF-β, Transforming growth factor-β; TNFα, Tumor necrosis factor-alpha.

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